Letters to the Editor

Diagnosing Environmental Allergy

With his commentary, "Total Environmental Allergy: 20th Century Disease or Deception?" (March 1990), Dr. Black has joined a small group of advocates who are using unscientific studies and tactics to deny the existence of an emerging, increasingly recognized public health problem. It is critical that your readers know the other side of the story before they, too, treat a growing group of difficult-to-treat patients as nut cases. Otherwise, the medical profession runs the risk of further damaging its credibility and doing a serious injustice to the victims of an illness that we admittedly do not clearly understand.

The first advocacy tactics employed in this article rest in the title and abstract. Why must we choose between "Disease or Deception"? Is there no room for uncertainty here? Are we so arrogant as to assert that we know everything about neuroimmune interfaces? Is there no room for definitive scientific research to finally put this matter to rest?

Dr. Black and his fellow advocates seek to discredit an emerging diagnosis. They are not pleading for mainstream research in this area; in fact, their articles serve to create an atmosphere in which serious scientists are afraid to engage in research for fear of career damage from being accused of supporting "clinical ecologists." This is no way to help unravel a truly difficult medical and societal concern.

Dr. Black also purports to explain the etiology of environmental illness, even though no claim has been made that a single etiology even exists. He sets up a straw man in "clinical ecology," failing to mention that multiple chemical sensitivity is a common diagnosis within occupational medicine and has far greater acceptance as a physiological illness in the medical community than does his theory of psychogenic illness. In fact, I was an invited participant at a 1987 workshop at the National Research Council during which Dr. Terr's psychogenic theory received no support. The clear consensus of that meeting was that this was a significant public health concern for which research must be supported and funded.

To be sure, Dr. Black and those he cites to support his psychiatric etiology theory have support. The Chemical Manufacturers Association, in its position paper,[1] has an almost identical perspective on this illness as Dr. Black. However, the papers by Terr and Stewart and Raskin are not scientific studies. They are merely case reviews which reflect the biases of the observers. Given an absence of any valid research design, none of them could have detected a physiological basis for this illness, even if one clearly existed. Terr's 50 cases,[2] it should be noted, were almost all cases referred to him by a workers' compensation insurance carrier, and for whom he failed to provide any control group or any testing protocol that could have met even the most rudimentary standards for research design.

However, even on its face, Dr. Black's article was not even-handed in his treatment of either this illness or *one* of the groups of physicians who deal with it, clinical ecologists. While criticizing the lack of research to support clinical ecology, Dr. Black has apparently based his opinions on one clinical vignette, a self-proclaimed obsessive-compulsive. In fact, nothing in his description of this patient even relates to environmental illness. Rather, the basic complaint appears to be chronic candidiasis. Yet, there is no proof that this condition has anything to do with the chemical sensitivity problem which is central to what Dr. Black terms "environmental illness."

In fact, it is difficult to ascertain exactly what Dr. Black is even talking about. It is clear that he has no respect for physicians he calls "clinical ecologists," but it is completely unclear whether he has any understanding of the standards of the American Academy of Environmental Medicine, the Pan American Allergy Society, and the American Academy of Otolaryngologic Allergy, all of which support the diagnosis and some of the attempts to aid these patients to whom Dr. Black refers.

When Dr. Black refers to "clinical ecology" he does not make it clear that he is referring to approved practices and techniques performed by a *Fellow* of the American Academy of Environ-

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mental Medicine or a *Diplomate* of the American Board of Environmental Medicine. No such physicians, according to the latest directories from these organizations, even exist in the State of Iowa. One must wonder whether Dr. Black's opinions are derived from a bad experience with someone who is not even representing clinical ecology. Either way, he does a disservice to your readers by exaggerating the "fringe" status of this medical perspective, omitting the fact that its courses have been granted CME accreditation.

To be sure, this entire field is confusing; however, Dr. Black only adds to that confusion. He provides incomplete references to mainstream articles that support the diagnosis, which appeared not in "Occupational Medicine" as in his references 1, 2, and 22, but rather, in "Occupational Medicine: State of the Art Reviews, Workers with Multiple Chemical Sensitivities," published by Hanley and Belfus, Philadelphia, 1987, and edited by Mark Cullen, M.D., Director of the Occupational Medicine Program at Yale. Clearly, the correct citation could have detracted from Dr. Black's position that this diagnosis is supported only by "fringe" medicine.

Dr. Black also failed to mention that the Ontario Ministry of Health Report he cites as reference 6 concluded that the environmental illness diagnosis was valid. He failed to mention that the U.S. Environmental Protection Agency (EPA) has recommended research on this illness,[3] that there is current research ongoing in this area at major medical centers, that two states have completed major reports on these illnesses,[4,5] and that the EPA and National Academy of Sciences are pursuing research in this area.

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and the class of the compound, carcinogenicity studies with ceftriaxone in animals have not been performed. The maximum duration of animal toxicity studies was six months. Mutagenesis: Genetic toxicology tests included the Ames test, a micronucleus test and a test for chromosomal aberrations in human lymphocytes cultured *in vitro* with ceftriaxone. Ceftriaxone showed no potential for mutagenic activity in these studies. Impairment of Fertility: Ceftriaxone produced no impairment of fertility when given intravenously to rats at daily doses up to 586 mg/kg/day, approximately 20 times the recommended clinical dose of 2 gm/day. *PREGNANCY:* Teratogenic Effects: Pregnancy Category B. Reproductive studies have been performed in mice and rats at doses up to 20 times the usual human dose and have no evidence of embryotoxicity, fetotoxicity or teratogenicity. In primates, no embryotoxicity or teratogenicity was demonstrated at a dose approximately three times the human dose. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproductive studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. Nonteratogenic Effects: In rats, in the Segment I (fertility and general reproduction) and Segment III (perinatal and postnatal) studies with intravenously administered ceftriaxone, no adverse effects were noted on various reproductive parameters during gestation and lactation, including postnatal growth, functional behavior and reproductive ability of the offspring, at doses of 586 mg/kg/day or less. *NURSING MOTHERS:* Low concentrations of ceftriaxone are excreted in human milk. Caution should be exercised when Rocephin is administered to a nursing woman. *PEDIATRIC USE:* Safety and effectiveness of Rocephin in neonates, infants and children have been established for the dosages described in the Dosage and Administration section. *In vitro* studies have shown ceftriaxone, like some other cephalosporins, can displace bilirubin from serum albumin. Rocephin should not be administered to hyperbilirubinemic neonates, especially prematures.
ADVERSE REACTIONS: Rocephin is generally well tolerated. In clinical trials, the following adverse reactions, which were considered to be related to Rocephin therapy or of uncertain etiology, were observed: *LOCAL REACTIONS*—pain, induration or tenderness at the site of injection (1%). Less frequently reported (less than 1%) was phlebitis after I.V. administration. *HYPERSENSITIVITY*—rash (1.7%). Less frequently reported (less than 1%) were pruritus, fever or chills. *HEMATOLOGIC*—eosinophilia (6%), thrombocytosis (5.1%) and leukopenia (2.1%). Less frequently reported (less than 1%) were anemia, neutropenia, lymphopenia, thrombocytopenia and prolongation of the prothrombin time. *GASTROINTESTINAL*—diarrhea (2.7%). Less frequently reported (less than 1%) were nausea or vomiting, and dysgeusia. Onset of pseudomembranous colitis symptoms may occur during or after antibiotic treatment (see WARNINGS). *HEPATIC*—elevations of SGOT (3.1%) or SGPT (3.3%). Less frequently reported (less than 1%) were elevations of alkaline phosphatase and bilirubin. *RENAL*—elevations of the BUN (1.2%). Less frequently reported (less than 1%) were elevations of creatinine and the presence of casts in the urine. *CENTRAL NERVOUS SYSTEM*—headache or dizziness were reported occasionally (less than 1%). *GENITOURINARY*—moniliasis or vaginitis were reported occasionally (less than 1%). *MISCELLANEOUS*—diaphoresis and flushing were reported occasionally (less than 1%). Other rarely observed adverse reactions (less than 0.1%) include leukocytosis, lymphocytosis, monocytosis, basophilia, a decrease in the prothrombin time, jaundice, gallbladder sludge, glycosuria, hematuria, anaphylaxis, bronchospasm, serum sickness, abdominal pain, colitis, flatulence, dyspepsia, palpitations and epistaxis

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Should we be relying upon the unscientific conjectures of Dr. Black, or should we withhold judgment until these studies are complete?

It is also unfortunate that Dr. Black failed to note that the 1981 position of the California Medical Association (CMA) has been withdrawn and is no longer in effect.[6] The article to which he refers in Ref. 24 is not the position of the CMA.

Should we look at the facts, or should we jump at the first easy explanations to come along? Certainly, it would be easy for us to just write off this entire illness as psychogenic and ship everyone off to a psychiatrist. But would that be right? Would it be ethical to assume that a new illness is psychogenic, without even ruling out a physiological basis? Is the "science" of Dr. Black any better than that which he rejects from "clinical ecologists"?

I am troubled by the "bandwagon" approach of anticlinical ecologists who want to throw out the baby with the bathwater. The patients are caught in a cross fire, and their interests are in no way served by untested theories which would serve to "blame the victim." I admit to being an advocate at times, but the difference is that I am advocating research, and Dr. Black is not. Also, I think we first must rule out physiologic illness before assuming psychogenesis, and Dr. Black thinks it has already been ruled out, or that his theory is so persuasive as to not require scientific verification.

It is troubling that physicians like Dr. Black can get away with attacking a diagnosis without scientific data or confirmed alternative diagnosis, or scientifically tested treatment protocol. Can anyone seriously claim that Terr's paper truly tested the hypotheses of clinical ecologists? Can it even be seriously claimed that clinical ecology theories should serve as the only basis for evaluating this illness?

In my experience, psychiatric approaches have not worked with confirmed cases of multiple chemical sensitivities, except to assist them in the tremendously stressful process of coping with this illness and with physicians and insurers who insist that the illness is "all in their heads." Psychogenesis is a diagnosis of last resort precisely because it defines away a problem that might, indeed, be real. In doing so, there is also a risk of immense harm to our economy and public health if this illness is, indeed, growing and is physiologically based and remains unrecognized.

I believe psychiatrists would describe this phenomenon as the need of the majority to "deny" any problem that makes them uncomfortable and challenges prevailing attitudes. This "denial" and the unscientific counter-attacks exemplified by Dr. Black, serve only to drive these patients further away from mainstream medicine and to vilify a group of physicians who attempt to make their lives more full and productive. Rather than developing a dialogue with those who have decades of experience with this illness, the tenor of the current debate is condemning and in no way conducive to good science.

There is room for disagreement in this area; however, the medical community deserves more evenhanded articles in this complex, emerging area of concern. Many, many mainstream physicians have seen this illness, and they need solid research, not generalizations and advocacy, to help them make their own decisions on this matter. For a thoughtful and complete review of this problem, I urge physicians to obtain a copy of the New Jersey studies mentioned above, which also somehow eluded mention by Dr. Black.

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References

1. "Environmental Illness" Briefing Paper, Chemical Manufacturers Association, Washington, D.C., 1989.
2. Terr AI: Environmental illness: A clinical review of 50 cases. Arch Int Med 146: 145-149, 1986.
3. U.S. Environmental Protection Agency Report to Congress on Indoor Air Quality. Washington, D.C., August 1989, p. 1.
4. Bascom R: Chemical hypersensitivity syndrome study. State of Maryland Department of the Environment, Baltimore, Maryland, March 1989.
5. Ashford NA, Miller CS: "Chemical sensitivity: A report to the New Jersey State Department of Health. Trenton, NJ, December 1989.
6. Meghrigian AG: California Medical Association Legal Counsel, April 9, 1990 (personal correspondence).

• *Dr. Black replies:*

Mr. Davis has set himself up as his own straw man. He has not challenged any of my fundamental points, nor has he provided any evidence to counter them. First, the diagnosis of multiple chemical hypersensitivity syndrome is unreliable; there are no adequate case definitions that would allow reasonable epidemiologic study. Second, the disorder is not associated with any reproducible laboratory abnormalities. Third, the tests that